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Assessment of Left Ventricular Function by Layered Strain in Hypertensive Patients with and without Left Ventricular Strain

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ABSTRACT

Background: ST depression and T-wave inversion (TWI) are classic ECG strain patterns that indicate left ventricular hypertrophy (LVH) and a poor prognosis. The relationship between strain and increased left ventricular (LV) mass and its relationship to ischemic heart disease (IHD), however, hasn't been thoroughly studied.

Objectives: The aim of the current work was to assess if hypertensive cases with a strain pattern on ECG have more ischemic changes at the level of the myocardium or if it is just an electrical phenomenon associated with hypertension (HTN).

Patients and methods: The current study comprised 100 hypertensive cases who were undergone coronary angiography for suspected angina pectoris and revealed normal coronaries, and 15 age and sex-matched normotensive health volunteers **(control group)**. Patients were further divided into 2 groups; **Group I:** included 50 hypertensive patients with strain criteria by ECG, and **Group II:** included 50 hypertensive patients without strain criteria. A conventional echocardiogram was performed using M-mode, 2D, Doppler, and Tissue Doppler, and then myocardial strain measured by 2D speckle tracking echocardiography (STE) was used to evaluate the layers of the myocardium.

Results: The layer-specific strain (LSS) was significantly lower in both hypertensive groups compared to the controls in all three layers (endocardium, myocardium, and epicardium). The LSS was significantly decreased in GI cases with strain ST-T changes than in GII cases with no strain ST-T changes. The endocardial layer was much more affected than the mid-myocardial and epicardial layers; in GI, the endocardial layer's P-value was <0.001, while in the mid-myocardial layer the P-value was <0.05), and in the epicardial layer P-value was <0.05.

Conclusion: It could be concluded that hypertensive patients with LVH and strain pattern have more ischemic changes than hypertensive patients without strain and the ischemic changes are more profound at the level of the endocardium.

Keywords: Hypertension, left ventricular strain, speckle tracking, layer-specific strain

INTRODUCTION

Stroke, atherosclerosis, and ischemic heart disease are only a few cardiovascular disorders for which HTN are a significant risk factor. A typical side effect of HTN is LVH, which has its own set of risks for cardiovascular morbidity and death [(1)].

A well-known indicator of anatomical LVH is the characteristic pattern of ST depression and TWI on the resting electrocardiogram (ECG) [(2)].

When ECG LVH criteria are employed for stratification, this anomaly of repolarization is the best predictor of poor outcomes. It has been linked to a poor prognosis in several clinical groups. Furthermore, in addition to the clinical effects directly linked to high left ventricular (LV) mass, the strain pattern could be a reflection of underlying coronary artery disease (CAD). This correlation may help to partially explain the clinical effects of this ECG finding [(3)].

A measurement of tissue deformation is strain. When the ventricles contract, the muscles undergo negative strain in the longitudinal and circumferential dimensions and positive strain in the radial direction. However, because global strain only evaluates global function rather than myocardial layer-specific activity, it cannot provide a thorough analysis of LV mechanics. The layer of the heart most susceptible to early injury from hypertension is the endocardium, but as the situation progresses, the pathology spreads and gradually impairs the function of the mid-myocardium and epicardium as well [(4, 5)].

As a result, different hypertension stages may cause layer-specific dysfunction that is difficult to identify with a single-layer evaluation. LSS is a novel technique that has the ability overcome such limitations; it permits an exhaustive evaluation of the three myocardial layers and, as a result, can determine the origins and evolution of myocardial mechanical dysfunction [(6)].

As a result, we aimed to assess if hypertensive cases with a strain pattern on ECG have more ischemic changes at the level of the myocardium or if it is just an electrical phenomenon associated with hypertension.

PATIENTS AND METHODS

This study included a total of 100 hypertensive patients who underwent coronary angiography for suspected angina pectoris and revealed normal coronaries, attending at Department of Cardiology, Menoufia University Hospital.

The included patients were recruited for the investigation of the effect of HTN on different layers of the myocardium using 2D speckle-tracking echocardiography.

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Inclusion criteria: Patients with chronic hypertension for a duration of more than 1 year, Sinus rhythm ECG, normal LV ejection fraction (EF>50%), and normal coronaries as proved by coronary angiography.

Exclusion criteria: Patients with moderate to severe valvular heart disease, any rhythm other than sinus rhythm, cardiomyopathies (dilated, hypertrophic and restrictive cardiomyopathy), congenital heart diseases, pericardial diseases, severe renal or hepatic diseases, and poor echocardiographic acoustic window.

The included subjects were divided into three groups; **Group I:** included 50 hypertensive patients with strain criteria by ECG, **Group II:** included 50 hypertensive patients without strain criteria and **Group III (control):** included 15 age and sex-matched normotensive health volunteers served as controls.

All the patients were subjected to:

1. A complete history was obtained from all cases, followed by a detailed clinical examination.
2. ***12-lead electrocardiogram (ECG):***
   To identify the beat and record LVH Paying close attention to the LVH-specific ECG voltage requirements, Sokolow-Lyon voltage requirements state that SV1+RV5 or RV6 ≥3.5 mV (35 mm) or R aVL ≥1.1 mV (11 mm) (respectively) [7, 8]. In a resting ECG, left ventricular strain is indicated by a downward-sloping asymmetrical ST-segment depression and an inverted asymmetric T wave ≥0.1mV opposite the QRS axis [9].
3. ***Trans-thoracic 2-dimensional (2D) Echocardiography:***
   Echocardiography was performed in the left lateral decubitus at end-expiration, in the parasternal long, short-axis, apical 2, 3, and 4-chamber views using a commercial ultrasound imaging system (GE Vivid S5 &Vivid E9 Ultrasound Machine) based on the recommendations of the ASE [10]**.** Measurements of chamber dimensions, wall motion abnormality, and valve morphology taken from 2D-guided M-mode. Doppler and Color Doppler for assessment of Valvular stenosis or regurg and the ratio of E/A. Septal mitral annular early diastolic (è) wave was measured by Tissue Doppler and the E/e' ratio was measured from early diastolic transmitral flow velocity (E) to mitral annular wave velocity (e').
4. ***2D-STE:***
   Three LV apical long-axis views were obtained; a) Apical 4-chambers, b) Apical 2-chambers c) Apical 3-chambers. At the end of the exhalation breath, three successive cardiac cycles were reported and digitally stored for offline assessment. Pulsed-wave Doppler measurements of LV inflow and outflow velocities were used to detect the onset of cardiac occasions. The endocardial, middle, and epicardial layers of the 2D images from the 3 apical views were included in the LSS, which was automatically generated for the investigation of longitudinal endocardial, mid-myocardial, and epicardial stresses [11, 12] as shown in figure (1).

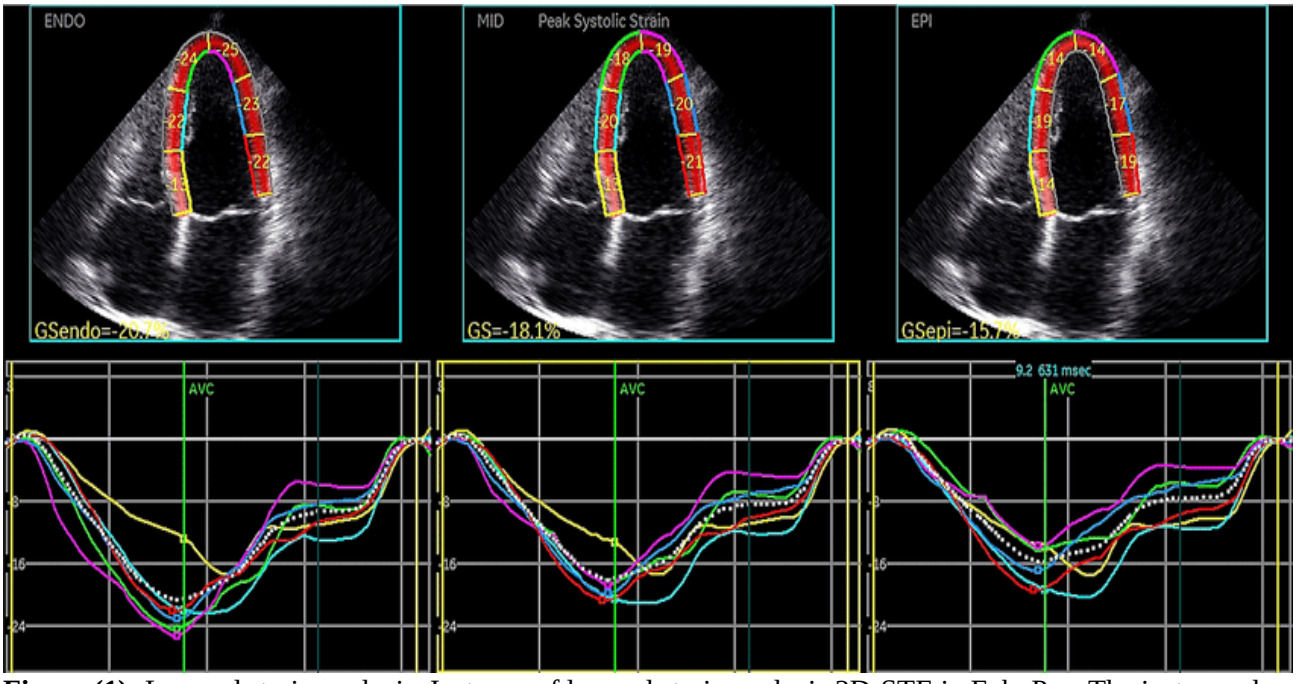

Figure (1): Layered strain analysis. Instance of layered strain analysis 2D-STE in EchoPac. The instance demonstrates STE in a four chamber view with assessment of the endocardial, whole wall (mid), and epicardial GLS [11-12]**.**

***Ethical Consideration*:**

This study was ethically approved by Menoufia University's Research Ethics Committee. Written informed consent of all the participants was obtained after being informed of the study's objectives and methodology. The study protocol conformed to the Helsinki Declaration, the ethical norm of the World Medical Association for human testing.

Statistical analysis

Version 20.0 of the IBM SPSS was utilized to appropriately evaluate the data after they were fed into the computer. The qualitative data were described by numbers and percentages. The Shapiro-Wilk test was applied to determine whether the distribution was normal. The quantitative data were defined by utilizing the mean±SD. The significance of the acquired results was detected at the 5% level. For pairwise comparisons, the Post Hoc test (Tukey), the F-test (ANOVA), and the Chi-square test for categorical variables were all employed for comparison between various groups. P values less than 0.05 were considered significant.

RESULTS

No statistically significant differences were recorded between all the studied groups as regard age, gender, and smoking. As regard diabetes, and dyslipidemia, there was a statistically significant difference between groups I and III, as well as between groups II and III, but no difference between groups I and II (as demonstrated in Table 1).

Table (1): Comparison between the 3 groups regarding demographics & risk factors.

	Group I Hypertensive patients with strain criteria by ECG (n = 50)		**Group II Hypertensive patients without strain (n = 50)**		**Group III Control group (n = 15)**		**Test of Sig.**	**Sig. bet. Groups. p**		
	Mean±SD		Mean±SD		Mean±SD			**I vs. II**	**I vs. III**	**II vs. III**
Age (years)	52.04 ± 4.55		50.20 ± 3.63		51.13 ± 3.54		F=2.579	>0.05	>0.05	>0.05
Sex	**No.**	**%**	**No.**	**%**	**No.**	**%**				
Male	30	60.0	24	48.0	**7**	46.7	χ^2= 1.727	>0.05	>0.05	>0.05
Female	20	40.0	26	52.0	8	53.3				
Smoking										
No	34	68.0	38	76.0	12	80.0	χ^2= 1.237	>0.05	>0.05	>0.05
Yes	16	32.0	12	24.0	3	20.0				
Diabetic										
No	27	54.0	28	56.0	15	100	χ^2= 11.454	0.841	0.001*	0.002*
Yes	23	46.0	22	44.0	0	0				
Dyslipidemia										
No	35	70	37	74	14	93	χ^2= 6.387	>0.05	0.04*	0.03*
Yes	15	30	13	26	1	7				

SD: Standard deviation **ECG**: electrocardiogram

P: p value for comparing among the three studied groups.

*: Statistically significant at $p \leq 0.05$

Patients on GI had a higher IVS, posterior wall thickness, and larger left atrium diameter compared to GII patients and GIII patients. In the context of LV mass and relative wall thickness, GI patients had larger LVM and RWT compared to GII patients and GIII patients (as demonstrated in Table 2).

Table (2): Comparison among the three studied groups Based on Echo parameters:

Echo parameters	Group I Hypertensive patients with strain (n = 50) Mean±SD	Group II Hypertensive patients without strain (n = 50) Mean±SD	Group III Control group (n = 15) Mean±SD	F	Sig. bet. Groups. p		
					I vs. II	I vs. III	II vs. III
IVSD (cm)	1.20 ± 0.13	1.11 ± 0.11	0.86 ± 0.14	46.23	<0.001*	<0.001*	0.002*
LVPWD (cm)	1.19 ± 0.13	1.0 ± 0.08	0.93 ± 0.11	54.37	<0.001*	<0.001*	<0.001*
LVEDD (cm)	4.88 ± 0.46	4.88 ± 0.34	4.79 ± 0.38	0.289	>0.05	>0.05	>0.05
LVESD (cm)	3.01 ± 0.32	3.13 ± 0.35	3.15 ± 0.37	2.060	>0.05	>0.05	>0.05
EF (%)	62.16 ± 2.62	62.18 ± 2.43	60.27 ± 5.86	2.391	>0.05	>0.05	>0.05
Aorta (cm)	3.45 ± 0.26	3.41 ± 0.19	3.33 ± 0.19	1.548	>0.05	>0.05	>0.05
Left atrium (cm)	3.98 ± 0.19	3.73 ± 0.19	3.51 ± 0.23	48.11	<0.001*	<0.001*	<0.001*
LVM (g)	205.17±42.85	169.90±46.17	138.19±32.15	84.64	<0.001*	<0.001*	<0.001*
RWT	0.61±0.10	0.52±0.08	0.37±0.03	50.11	<0.001*	<0.001*	<0.001*
E wave (m/s)	0.59 ± 0.15	0.67 ± 0.10	0.75 ± 0.12	5.859	0.004*	0.012*	0.042*
A wave (m/s)	0.81 ± 0.14	0.74 ± 0.08	0.71 ± 0.17	5.489	0.005*	0.023*	0.019*
E/A ratio	0.74 ± 0.18	0.91 ± 0.16	1.05 ± 0.35	16.91	<0.001*	<0.001*	<0.001*
e'(m/s)	0.04±0.02	0.06±0.02	0.09±0.03	18.31	<0.001*	<0.001*	<0.001*
E/e' ratio	14.17 ± 1.63	11.57 ± 1.67	6.26 ± 1.26	40.49	<0.001*	<0.001*	<0.001*
EPASP	24.60 ± 5.33	22.70 ± 3.53	24.0 ± 5.41	2.132	>0.05	>0.05	>0.05

IVSD: **interventricular septum diameter;** LV**PWD**: left ventricular posterior wall diameter; LVEDD: **left ventricular end-diastolic diameter;** LVESD: **left ventricular end-systolic diameter;** EF: **ejection fraction;** LVM: **left ventricular mass;** RWT: **relative wall Thickness;** EPASP**:** estimated pulmonary artery pressure**. F:** F for One way ANOVA test**,** Pairwise comparison between each 2 groups was done using Post Hoc Test (Tukey)**, SD**: Standard deviation**. P**: p-value for comparing between the three studied groups. ***:** Statistically significant at $p \leq 0.05$

More LV diastolic function affection in group 1 in comparison to group 2 and controls as evidenced by a decrease in the (E) wave and elevation in the (A) wave, also E/A ratio was lower in GI compared to GII and GIII and E/e' ratio was higher in GI compared to GII and GIII.

Regarding the LSS, it has been demonstrated to be associated with a significant reduction in both hypertensive groups in comparison to the controls in all the 3 layers (endocardium, myocardium, and epicardium). The LSS was significantly reduced in GI cases with strain ST-T changes than in GII cases with no strain ST-T changes. The endocardial layer was much more affected than the mid-myocardial and epicardial layer, the endocardial layer in GI P value was <0.001while in the mid-myocardial layer P value was <0.05, and in the epicardial layer P value was <0.05. This was similar in the apical four-chamber, 3-chamber, and two-chamber views as shown in Table (3). Example cases of patients with and without strain are shown in Figures (2, 3, and 4)**.**

Table (3): Comparison among the three studied groups according to Layer specific strains in 4CH, 2CH and 3CH view:

	Group I Hypertensive patients with strain criteria by ECG (n = 50) Mean±SD	Group II Hypertensive patients without strain (n = 50) Mean±SD	Group III Control group (n = 15) Mean±SD	F	Sig. bet. Groups. p		
					I vs. II	I vs. III	II vs. III
Endocardium 4CH	-16.26 ± 1.85	-18.80 ± 1.83	-21.75 ± 1.90	20.245*	<0.001*	<0.001*	<0.001*
Endocardium 2CH	-15.54 ± 2.26	-18.56 ± 2.05	-19.29 ± 2.00	87.363*	<0.001*	<0.001*	<0.001*
Endocardium 3CH	-16.06 ± 1.92	-17.24 ± 2.17	-18.65 ± 2.68	14.074*	<0.001*	<0.001*	<0.001*
Myocardium 4CH	-16.97 ± 1.64	-17.68 ± 2.88	-18.27 ± 2.63	6.579*	<0.05*	<0.05*	<0.05*
Myocardium 2CH	-16.85 ± 1.89	-17.54 ± 1.92	-18.29 ± 1.71	6.905*	<0.05*	<0.05*	<0.05*
Myocardium 3CH	-14.96 ± 1.91	-15.72 ± 2.28	-16.49 ± 2.02	8.067*	<0.05*	<0.05*	<0.05*
Epicardium 4CH	-15.88 ± 2.73	-16.59 ± 2.88	-17.29 ± 1.78	6.689*	<0.05*	<0.05*	<0.05*
Epicardium 2CH	-15.77 ± 2.57	-16.47 ± 1.95	-17.09 ± 1.88	7.131*	<0.05*	<0.05*	<0.05*
Epicardium 3CH	-13.43 ± 2.10	-14.61 ± 2.63	-15.69 ± 2.76	8.869*	<0.05*	<0.05*	<0.05*

SD: Standard deviation **ECG**: electrocardiogram **F:** F for One way ANOVA test**,** Pairwise comparison between each 2 groups was done using Post Hoc Test (Tukey) **P**: p-value for comparing between the three studied groups.*: Statistically significant at $p \leq 0.05$

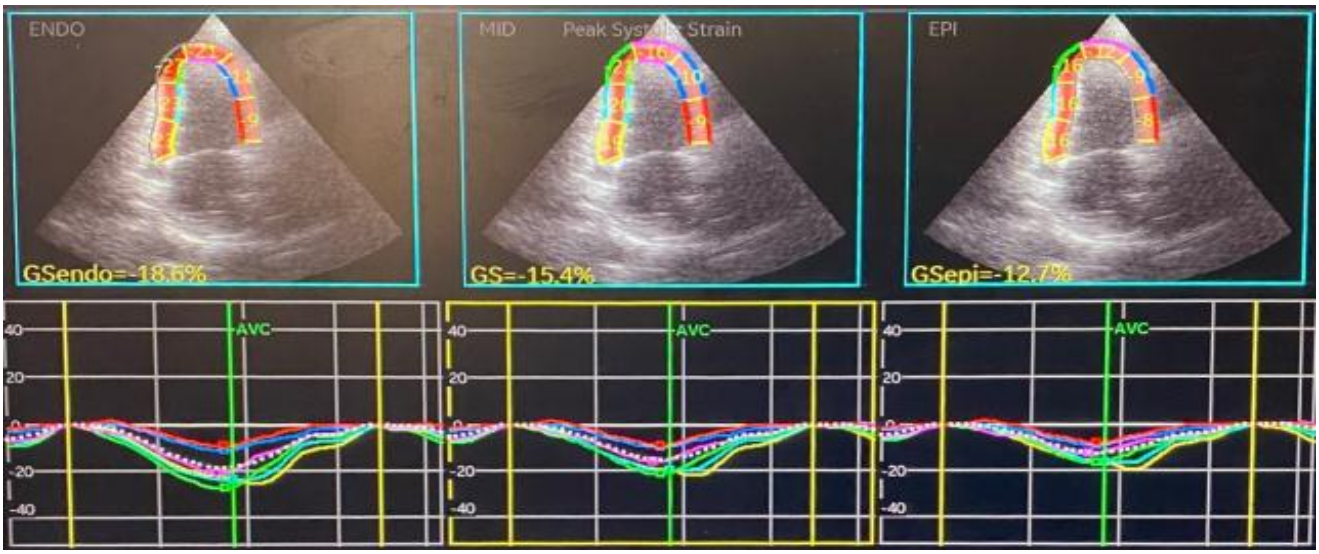

Figure (2): 2D-STE measurement of global LSS in apical 2-chamber view in a hypertensive patient with strain, the quantitative strain measurements of the endocardium GSendo (-18.6), middle GSmid (-15.4), and epicardium GSepi (-12.7).

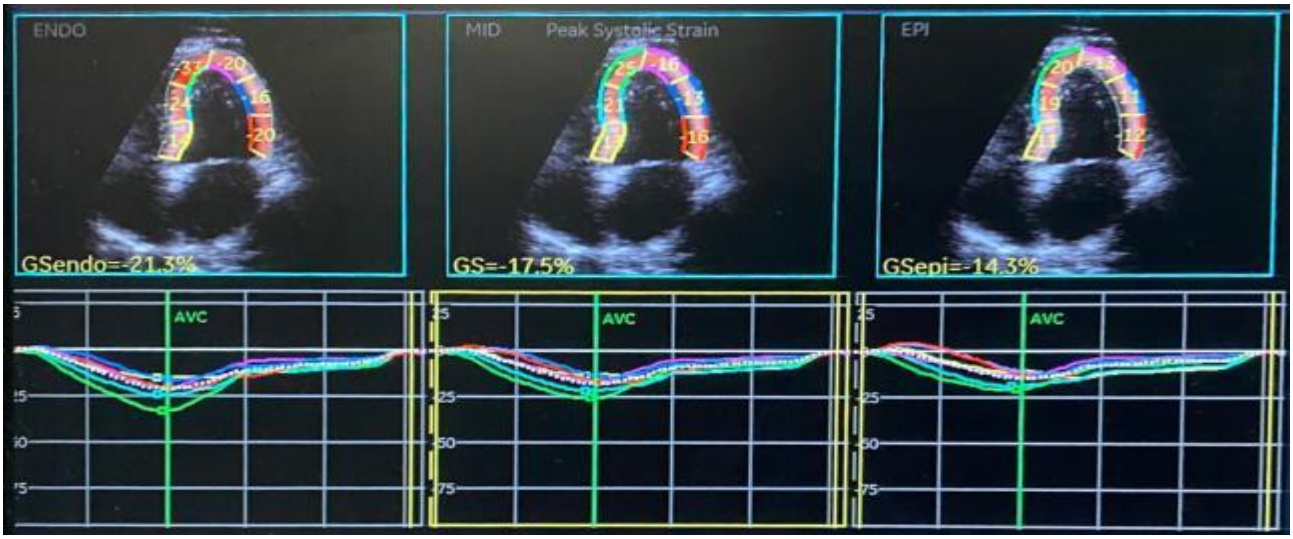

Figure (3): 2D-STE measurement of global LSS in apical 4-chamber view in a hypertensive case with no strain, the quantitative strain measurements of the endocardium GSendo (-21.3), middle GSmid (-17.5), and epicardium GSepi (-14.3).

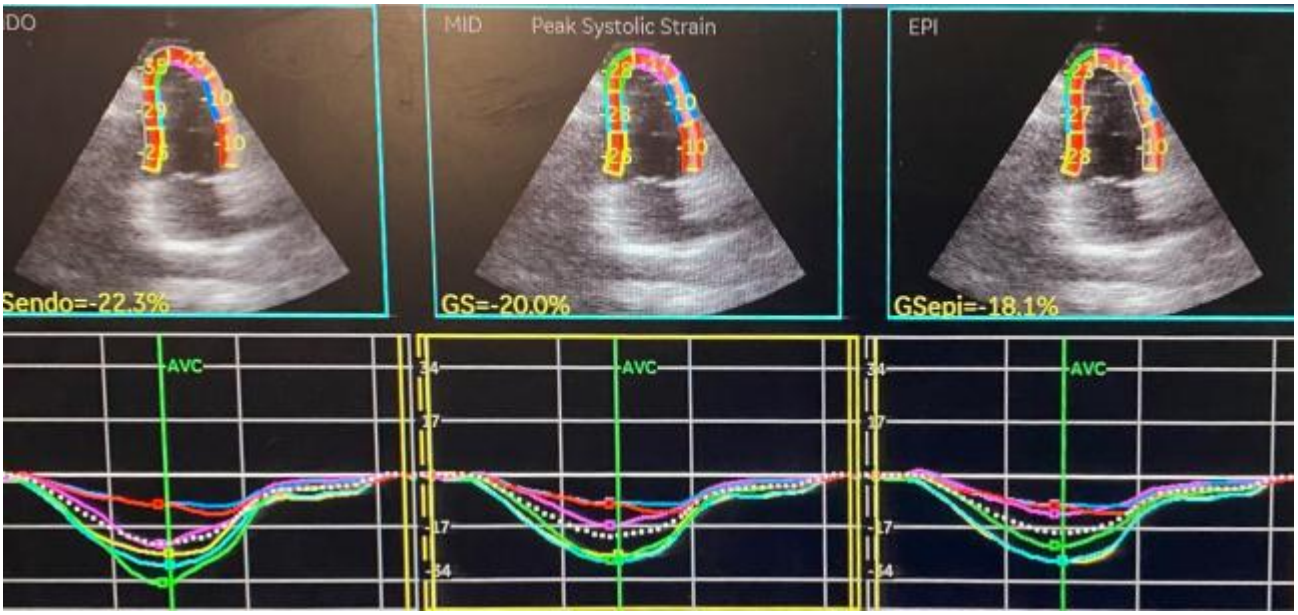

Figure (4): 2D-STE measurement of global LSS in apical 2-chamber view in a normotevsive individual (control group), the quantitative strain measurements of the endocardium GSendo (-22.3), middle GSmid (-20.0), and epicardium GSepi (-18.1).

DISCUSSION

The current study concluded that, in the context of both hypertension groups compared to the control group, the LSS was lower in all three layers of the myocardium (endocardium, mid-myocardium, and epicardium). When comparing group I, cases with strain ST-T changes with group II patients without strain ST-T changes, it was more pronounced in the endocardial layer than the mid-myocardial and the epicardial layers. An observation that suggests that subendocardial dysfunction is reflected in strain ST-T alterations.

Early signs of cardiac problems were often seen in the subendocardial layers owing to particular properties of coronary perfusion and myocardial requirement. Epicardial vessels and intramural vessels were the two categories used to describe coronary arteries. The former served as conductors of blood flow and were bigger and more superficial. These later vessels were smaller and more course inside the myocardium; they offer more resistance but finer regulation of blood flow thanks to their many branches and arterioles. During ventricular systole, blood flow in the majority of tissues reaches its peak because of increased pressure in the aorta and its distal branches. Blood flow through the coronary vessels, on the other hand, appears to be paradoxical, peaking during ventricular diastole. This unique pattern results from the external compression of coronary arteries by cardiac tissue during systole. The vessels in the endocardial layer experience the greatest compressive force, while the vessels in the epicardium experience the least. Overall, this causes lower coronary flow due to increasing ventricular pressure and higher oxygen demand as a result of growing muscle mass [(13)].

In previous 2D speckle-tracking experiment, **Takeuchi *et al.*** [(14)] showed that hypertensive cases with normal LVEF had less longitudinal strain.

Similar results were reached by **Shalaby *et al.*** [(15)], a total of 50 hypertensive patients—25 with LVH and 25 without LVH—were included in the study, along with a 25-person age- and sex-matched control group. Researchers discovered that both groups of hypertensive cases in presence or absence of LVH showed significantly lower global longitudinal LV systolic strain than the controls.

The present observation was in agreement with **Kim *et al.*** [(16)] who studied 145 patients (61 ± 12 years) with primary hypertension and thirty one normotensive controls (63 ± 9 years) were prospectively comprised. Despite the fact that global LS did not change between the hypertensive and control groups, their investigation discovered that longitudinal strain at the endocardium and longitudinal strain at the mid-myocardium were lower in cases with hypertension but no LVH compared to the controls (both P 0.05).

Other causes of LVH, such as left ventricular outflow obstruction, as in aortic stenosis, showed similar affection to LSS. The effect of AS on cardiac functions as determined by utilizing 2D-STE was evaluated in cases with aortic valve stenosis. Left ventricular hypertrophy had been linked to layered strain worsening, which could be caused by both hypertension and AS. A study by **Ilardi *et al.*** [(17)] which was conducted on 249 patients with severe AS, all myocardial layers were affected by longitudinal strain impairment, but the endocardial layer was the most noticeable. As the disease progresses, this disability was progressively more obvious.

An increase in QRS complex amplitude, increase in QRS complex length, ST segment depression, and a TWI were the key ECG-derived diagnostic features for LVH diagnosis. Additionally, it had been shown that alterations in QRS amplitude and shape reliable with ECG LVH criteria result from regional reduction of conduction velocity in the LV. An altered QRS complex that matched the diagnostic ECG LVH criteria or the QRS patterns often observed in LVH patients was caused by a decreased conduction velocity in the anteroseptal region of the LV. The lengthening of the QRS is assumed to be caused by a slowing of conduction velocity. Contrarily, the geometry of the activation front and, as a result, the size and direction of the depolarization vectors are impacted by diffuse or regionally reduced conduction velocity in the LV [(18)].

In addition to the voltage requirement for ECG-LVH, ECG LVH with strain pattern was described as the presence of a down-sloping asymmetrical ST-segment depression and an asymmetric TWI 0.1mV opposite the QRS axis in a resting ECG. An ST-segment depression bent upward and descends down into an asymmetrical TWI was a typical definition of left ventricular strain. Several clinical diseases are accompanied by a bad prognosis when the usual ECG strain pattern is present, making it a well-known indication of anatomical LVH. When ECG LVH criteria are used in the context of risk stratification, ST-segment depression and TWI are recognized as the greatest indication of morbimortality. It has been established that the significant link between strain on the ECG and rising LV mass is independent of coronary artery disease, despite the possibility that the ECG strain pattern possibly reflects the existence of underlying CAD [(19)].

The dysfunction of vascular smooth muscle cells and endothelial cells in HTN has been demonstrated to be associated with an increase in the stiffness of both major and minor blood arteries. In addition, HTN is characterized by a high PWV that could lead to pressure wave reflection in the cardiac microcirculation and possibly alter all three layers of the myocardium [(20)].

Limitations of the study:

First, the study was conducted on a small scale of cases, which may have had an influence on the results and diabetic cases were comprised in the study due to the limited number of cases that underwent coronary angiography without associated risk factors. Second, in order to acquire the best delineation of the endocardial boundary, speckle-tracking techniques tightly rely on the frame rate and high-quality 2-dimensional pictures. Third, recruited subjects had different onset, duration, and severity of hypertension. Finally, all measurements and examinations were performed while patients were under their anti-hypertensive medication.

CONCLUSION

It could be concluded that hypertensive patients with LVH and strain pattern have more ischemic changes than hypertensive patients without strain and the ischemic changes are more profound at the level of the endocardium.

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